Cardiac effects of anabolic steroids

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Anabolic steroid abuse in athletes has been associated with a wide range of adverse conditions, including hypogonadism, testicular atrophy, impaired spermatogenesis, gynaecomastia, and psychiatric disturbance. But what effect does steroid abuse have on the cardiovascular system?

Exogenously administered steroids

Given these putative effects of steroid hormones (and AAS in particular) on LV growth, we might expect exposure to exogenously administered steroid hormones to be associated with an exaggerated LV hypertrophic response to any other hypertrophic stimulus.

Exercise is just such a potent cardiac hypertrophic stimulus. Meanwhile, athletes are increasingly exposing themselves to supra-physiological doses of AAS. These are known to increase skeletal muscle mass and strength—effects which form the basis for their administration to enhance athletic performance. A variety of AAS are often taken simultaneously (so called “stacking”), and in doses which result in 10–100 fold increases in androgen concentrations. Administration regimens usually involve a 6–12 week cycle and are often administered in a “pyramidal” fashion, with doses tapering from low to high to low. Abused substances include testosterone, its 17-β esters, and those based on modified steroid rings (including 17-α derivatives).

The largest group to make such use of AAS are the very group whose LVH response to exercise is likely to be the greatest—the strength or resistance training (RT) athletes. One study from 1995 suggested that two thirds of elite US powerlifters have self reported use of AAS to enhance performance; even “dope testing” may be underestimating the true extent of such use. What evidence is there that AAS administration enhances the LV hypertrophic response to resistance exercise?

In this issue of Heart, Urhausen and colleagues report the results of a cross sectional study of cardiac morphology in relation to AAS use. Male bodybuilders/powerlifters currently using AAS or ex-users who had abstained from AAS exposure for over 12 months (U and ExU, n = 17 and 15, respectively) were compared to 15 weightlifters who denied current or past use of AAS (WL). Left ventricular wall thickness and cavity dimensions were assessed using echocardiography, and muscle mass (LVMM) calculated using the Devereux equation. Absolute LVMM measures (mean (SD)) were significantly greater for U than ExU or WL (281 (54) g v 232 (42) g v 204 (44) g for U v ExU v WL, respectively), with differences between ExU and WL only reaching significance after adjustment for body surface area or fat-free mass. These results suggest that AAS use increases the LV hypertrophic response.

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Abbreviations: AAS, anabolic/androgenic steroids; AR, androgen receptor; ExU, ex-users of anabolic steroids; hCG, human chorionic gonadotrophin; hGH, human growth hormone; LVH, left ventricular hypertrophy; LVMM, left ventricular muscle mass; RAS, renin-angiotensin system; RT, resistance training; U, users of anabolic steroids; WL, weightlifters
to exercise, an effect which might last for well over a year.

**CAUTION NEEDED**

Such data must nonetheless be treated with caution. We know, for example, that the magnitude and pattern of hypertrophy is dependent on the nature, duration, and intensity of exercise undertaken. Thus, strength trained athletes (such as weightlifters, powerlifters, bodybuilders, and throwers) develop a greater increase in wall thickness, a more concentric pattern of LV growth, and a lesser increase in athletes (such as weightlifters, powerlifters, bodybuilders, and throwers) which cardiac growth and cardiovascular disease are mediated directly, or through secondary phenotypes representing a 38–50% rise since 1991. The influence of steroid hormones on the heart thus warrants further study. Evidently, the potential impact of steroid abuse on public health is a matter of concern. Perhaps more importantly, however, such studies might lead to a greater understanding of the shared mechanisms through which cardiac growth and cardiovascular disease are mediated. Such issues are increasingly exciting as the identification of local myocardial steroid synthesis (and its potential pathogenicity) is paralleled by the demonstrated efficacy of steroid antagonists in cardiac disease. We might yet see a role for steroid antagonists such as aldosterone in the primary or secondary prevention of LVH, and its associated cardiovascular sequelae.

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**REFERENCES**

Images in Cardiology

EX filter wire usage in stenting right coronary artery lesion with diffuse aneurysmal dilatation

A 63 year old man was transferred to our hospital in an emergency following the onset of chest pain two hours previously. The ECG showed normal sinus rhythm with ST segment elevation in the anterolateral leads. He had a history of active Crohn’s disease.

An urgent cardiac catheterisation revealed a 99% mid-left anterior descending artery (LAD) stenosis (culprit lesion) and a 99% stenosis of an aneurysmal right coronary artery (RCA) (panel A). It was decided to attempt percutaneous coronary intervention (PCI). A glycoprotein IIb/IIIa inhibitor was not administered because of the bleeding risk related to Crohn’s disease. The LAD lesion was predilated and a stent 2.75 mm × 33 mm (Cypher, Cordis) was implanted with a TIMI grade 3 flow. Subsequently it was decided to treat the RCA stenosis. In order to avoid distal embolisation from mural thrombus within the ectatic segment, we opted to use a distal protection device (Filter Wire EX, Boston Scientific). Until now distal protection systems have been mainly used in the setting of acute coronary syndromes, and to treat thrombotic or degenerated saphenous vein graft stenosis.

After positioning the distal protection device, the RCA lesion was predilated with a 4.0 × 20 mm Maverik balloon at 12 atm. A 5.0 × 18 mm Express stent was then deployed at 16 atm. A massive embolisation occurred during angioplasty and stent implantation, resulting in a partial occlusion of the filter system but without distal embolisation (panel B). When the filter was retrieved a TIMI grade 3 distal flow was achieved and the no-reflow phenomenon did not occur (panel C). Inspection of the filter revealed the presence of many particles (panel D) which, at histological examination, appeared to be composed of fibrin, cholesterol crystal, foam cells, and amorphous material. These findings suggest that a large piece of intimal debris peeled off and was dislodged from the main plaque during the interventional procedure. The patient’s subsequent clinical course was uncomplicated.

The use of distal protection devices in the treatment of an RCA lesion within a diffuse ectatic segment, even in the angiographic absence of apparent thrombus, is suggested.

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